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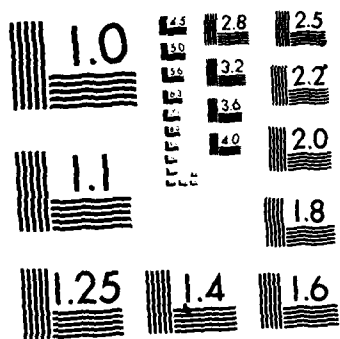
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EFFECTS OF DIETARY SODIUM ON MUSCLE WATER
AND ELECTROLYTES DURING HEAT ACCLIMATIZATION

ANNUAL SUMMARY REPORT

AUTHORS: David L. Costill, Ph.D.
Lawrence E. Armstrong, Ph.D.

DATE: April 30, 1984

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HUMAN PERFORMANCE LABORATORY
BALL STATE UNIVERSITY
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temperatures, and a smaller plasma volume expansion than during the HI regimen. Though plasma K⁺ concentration was unchanged throughout both treatments, the plasma K⁺, Na⁺ and Cl⁻ content increased in proportion to the increase in plasma volume. Muscle water, Na⁺, and Cl⁻ contents were significantly increased ($P < .05$) as a consequence of the heat acclimatization period, but no differences ($P > .05$) were found between the two Na⁺ diets. Muscle K⁺, on the other hand, remained unaffected by either the days of heat-exercise stress or the HI-LO diets. Although the LO diet appeared to lessen the subjects' acclimatization to the heat, neither diet affected the body K⁺ content, nor did they effect the excretion of K⁺ in urine or sweat.

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SUMMARY

This annual summary of research will report only the first of two major projects conducted during the first year of this contract period. The study reported here deals only with the influence of diets high and low in sodium, and their effect on heat acclimation and body potassium balance. The second investigation examines the effects of heat acclimation on endurance (aerobic) and sprint (anaerobic) exercise performance. This study has only recently been completed and the tissue (muscle and blood) samples are still being analyzed. For that reason the study will be included with the first quarterly report and the second annual (1984-85) report.

PROJECT I.

It has been suggested that renal conservation of Na⁺ during training in hot environments results in K⁺ deficiencies. This investigation examined the influence of two levels of dietary Na⁺ (399 vs 98 mEq Na⁺/day) on intramuscular, urinary, sweat, and plasma K⁺ homeostasis. Both diets included 80mEq K⁺/day. Nine unacclimatized, untrained males underwent successful acclimation during two 8-day regimens (40.1±.1°C, 23.5±.4 %RH). The low sodium diet was associated with higher heart rate (p < .05, D3-D5), higher rectal temperature (p < .05, D3-D6), lower sweat responsiveness, and delayed plasma volume expansion (P < .05, D4). Daily plasma K⁺ concentrations were stable, and total plasma K⁺, Na⁺, Cl⁻ increased is-osmotically with plasma volume (D4). Both diets resulted in depressed urinary K⁺ excretion. Sweat K⁺ and muscle K⁺ concentrations were not altered by diets or acclimation. Whole-body K⁺ balance responded similarly to both diets (+4.1%, +3.4%). This dietary-acclimation protocol did not result in whole-body K⁺ deficits.

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FOREWORD

For the protection of human subjects the investigators have adhered to policies of applicable Federal Law 45CFR46.

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Introduction

Dehydration and large sweat losses have been shown to limit physical performance (1,2,3,4) and to impair the body's controls on thermoregulation (5,6). Electrolyte losses in sweat and urine also have been suggested to affect the contractile characteristic of muscle (7,8), metabolism (9), and resistance to fatigue (9,10).

The importance of potassium (K⁺) in cellular metabolism is well established. Some investigations have noted potassium deficiencies in men exposed to several days of hard exercise in the heat (11,12). In contrast, no potassium deficit was observed when the effects of low dietary potassium intake were measured during repeated days of heavy exercise and sweating (13). In light of the important role played by K⁺ in neuromuscular conduction and glycogen synthesis, the present study was designed to determine the effects of dietary Na⁺ on the muscle and blood K⁺ contents of men during repeated days of exercise in the heat.

Previous studies which have examined dehydration and potassium balance reported changes in plasma, sweat and urine water and electrolytes (14,15,16). More recently we (13) have measured the effects of successive days of exercise in the heat on intramuscular potassium contents. The intent of this investigation was to examine the influence of dietary sodium intake on intramuscular and extracellular potassium contents of unacclimatized men who performed daily exercise in a hot environment. This investigation attempted to provide information regarding the physiological changes or debilitating effects of thermal stress on persons who are suddenly exposed to hot environments. Individuals subject to such environments include those involved in military positions, physical fitness training, and many occupations (e.g. farmers, steel workers, and construction workers).

Materials and Methods

Nine college-aged men were selected from a group of volunteers, after undergoing a physical examination and a treadmill maximal oxygen consumption test. The exercise test and a 30 day activity recall were used to verify that each subject was not involved in regular physical activity. The subjects were informed of all risks and stresses associated with this investigation before giving their written consent to participate. Their pertinent physical characteristics appear in Table I.

All subjects participated in two dietary-acclimation treatments which lasted 8 days each. In one 8 day sequence (High Na⁺ diet) the subjects ate a diet containing 399 mEq Na⁺/day and 80 mEq K⁺/day. The other 8 day sequence (low Na⁺ diet) required subjects to eat 98 mEq Na⁺/day and 80 mEq K⁺/day. The order of the treatments was randomized in a cross-over design (Figure 1) so that half of the subjects consumed the high sodium diet during the first sequence and the other half consumed the low Na⁺ diet

first. An average of 24.1 ± 1.7 days (mean \pm SE) elapsed between the two treatments. All food consumed by the subjects was provided for them. This involved three primary meals and one evening snack (approx. 3000 Kcal/day). In addition to the liquids supplied by these diets, subjects were allowed to consume tap water ad libitum, containing 0.5 mEq Na⁺/l and 0.3 mEq K⁺/l.

Urine collection commenced with the initial meal of each treatment diet. Subjects carried clean inert containers during their daily business and returned these containers to the laboratory every 24 hours. Two days of control urine output (9 subjects, n=18) were collected prior to the second treatment. Urine volume and the concentration of urine electrolytes were used to calculate the total daily loss of electrolytes in urine (mEq/24 hr). Loss of electrolytes in feces was not measured, but previous research has shown that this loss is less than 1.5 mEq Na⁺/day and 4 mEq K⁺/day (17).

The mean outdoor dry bulb temperatures appear in Figure 1. All acclimation trials were conducted between March and early May. Based on the 30 day activity recall forms and the mean daily outdoor temperatures, all subjects were classified as unacclimatized prior to the first treatment.

Subjects walked on a motor driven treadmill for 90 minutes on 8 consecutive days, during each dietary regimen. Treadmill velocity was calibrated at 5.6km/hr and 6% grade prior to the investigation and at regular dates thereafter. This exercise protocol previously had been demonstrated to induce acclimation successfully (18). The studies were conducted in an environmental chamber, maintained at 40.1 (SE ± 0.1)°C and 23.5 (SE ± 0.4) % RH. Air flow across the head and chest was measured at 0.92 m/sec using a hand held anemometer (FloRite).

During each acclimation trial, the following physiological measurements were taken. Skin temperatures were recorded from thermistors placed on the lateral upper arm, abdomen, and antero-lateral thigh (YSI, Yellow Springs, Ohio). Rectal temperature was recorded from a probe inserted to a depth of 8 cm beyond the anal sphincter. Mean skin temperature and mean body temperature were calculated using previously described formulae (19,20). Nude body weight was recorded (± 50 g) prior to and immediately following each trial. Subjects were allowed to drink water ad libitum during all exercise bouts, and the volume of water consumed was added to the body weight change to determine total sweat loss. Heart rates were recorded at 10 minute intervals using a portable cardiometer (Respiromics, Monroeville, PA). Respiratory exchange gases were collected in Douglas bags at rest, 15, 60, and 90 minutes of each trial, and were analyzed for oxygen consumption. Exercise was terminated if heart rate exceeded 180 beats per minute or if rectal temperature exceeded 39°C. During the first treatment, 8 out of 72 trials were terminated prior to 90 minutes; these reduced times were duplicated during the second treatment.

Four electrolytes were measured in all physiological samples: Na⁺, K⁺, Cl⁻, and Mg⁺⁺. Instrumentation and techniques have been described elsewhere (20). Figure 2 illustrates the schedule used for the collection of samples. All blood, urine and sweat assays were done in duplicate, whereas every muscle assay was done in triplicate.

Sweat electrolytes were collected following the first, fourth, and eighth work bouts using the whole body and clothing washdown technique described by Vellar (21). Subjects began trials with shorts, socks and a towel which were double-rinsed daily. During exercise bouts, subjects blotted dripping sweat from their bodies with an electrolyte-free towel. After washing subjects, clothing and towels with a known volume of deionized water, the total loss of electrolytes in sweat and the initial sweat concentration were calculated. Mean sweat rate per hour (ml/hr·m²) was determined using weight losses during acclimation trials. These sweat rates were corrected for water ingestion, metabolic water loss, and surface area differences between subjects. Sweat responsiveness was expressed per degree of rectal temperature change (ml/hr/°C).

Subjects reported for muscle and blood sampling in a 12 hour postprandial state. The initial blood sample was taken without stasis, following a minimum of 20 minutes of lying supine. Hemoglobin was measured by the cyanmethemoglobin method (Hycel, Inc., Houston, TX). The micro-hematocrit technique was used without correction for trapped plasma; total plasma proteins were analyzed using the biuret method (20). Total plasma osmolality was determined by freezing point depression (Advanced Instruments, Needham Heights, MA). Day 1 (control) plasma volumes were measured by Evans blue dye (T-1824) dilutions which had circulated in the vasculature for exactly 10 minutes. Relative changes in plasma volume (D4 and D9) were determined from changes in hematocrit and hemoglobin (22,23). Red blood cell volume was assumed to remain constant during acclimation trials (24).

The muscle biopsy technique of Bergstrom (25) was modified to include suction (26). Immediately after opening the needle containing the sample a stopwatch was started. Samples were cleaned of all visible fat and connective tissue, and were weighed 3-4 times at 15-30 second intervals. A linear regression equation representing water evaporation was used to extrapolate the zero second wet weight. After weighing, samples were dried at 80°C for two hours and placed in capped test tubes. Again, the samples were weighed to obtain dry weight and were immersed in 10ml of petroleum ether to extract all lipids. After one hour of lipid extraction, the samples were dried at 80°C for 30 minutes and reweighed to obtain fat-free solid weight. Muscle electrolytes were extracted in 200-300ul of 2N HNO₃ overnight.

To test for significant differences, a two-way analysis of variance (0.05 level of confidence) and the Duncan's NMR post hoc analysis were used. The two factors in this design were: (a) diet (two levels - low Na⁺ and high Na⁺), and (b) days (three levels - D1, D4, D9). Urine values and physiological measurements taken during acclimation trials included eight levels of the day factor (D1 through D8). All means are reported \pm SE.

Results

During a 48 hour period immediately prior to participating in this investigation, subjects consumed an average of 123.8 (\pm 18.2) mEq Na⁺/day, 82.1 (\pm 12.1) mEq K⁺/day, and 2310 (\pm 233) Kcal/day. Nine hundred Kcal were added to both treatment diets to compensate for energy expended during daily acclimation trials. The treatment diets proved to be good approximations of preinvestigation diets, with respect to K⁺ content and calories. The low Na⁺ diet supplied 26 mEq Na⁺/day less--while the high Na⁺ diet supplied 275 mEq Na⁺/day more--than the average preinvestigation diet.

During the eight days of exercise in the heat, the daily pre-trial mean body weights of subjects were not significantly affected by dietary manipulation or by acclimation ($p > 0.05$). Figure 3 illustrates mean body weight data, heart rate at 90 min of exercise, rectal temperature, and oxygen consumption data. Statistically significant differences in mean heart rate and mean rectal temperature (when compared to D1) were seen from D3 through D8 of acclimation trials. Although mean heart rates and rectal temperatures were lower during the high Na⁺ diet, significant differences existed between the diets on D3-D5 (heart rate) and D3-D6 (core body temperature) only. The daily exercise performed by subjects required a mean consumption of 1.74 (\pm 0.03) liters O₂/min (44.6-49.7% of maximal oxygen uptake), at the end of 90 minutes of work. Neither dietary treatments nor acclimation trials resulted in oxygen consumption differences.

Table II describes sweat rate and sweat responsiveness results for both diets. Sweat production rate exhibited no significant between-group or within-group differences. During the low Na⁺ diet, mean sweat responsiveness was lower for six out of the eight trials, but only D2 values were significantly different. Mean sweat electrolyte losses are presented in Tables IIIa and IIIb. Potassium losses in sweat were not significantly affected by dietary intervention, and did not change during acclimation trials. Magnesium responded in a similar pattern. The extracellular electrolytes sodium and chloride responded to this investigatory design in harmony, as well. Both sodium and chloride appeared in significantly lower concentrations while subjects were consuming the low Na⁺ diet (D4 and D8). During acclimation trials, the high Na⁺ diet resulted in elevated sweat losses of both ions, while the low Na⁺ diet resulted in significantly depressed sodium and chloride losses.

Mean daily urine volume (Tables IVa and IVb) was characterized by large within-diet fluctuations ($>600\text{ml}$), in which the low Na^+ diet and the high Na^+ diet were different on D5 only. The possibility of a type I statistical error is recognized. Interestingly, two distinct trends were seen during acclimation trials. The high Na^+ diet was characterized by a significant urine volume decrease on D1 and normal urine outputs on all days which followed. The low Na^+ diet, in contrast, was characterized by a daily decrease in mean urine volume through D4, at which time it increased to a level nearly identical to the D1 value. Urine electrolyte losses also are reported in Tables IVa and IVb. As was seen with sweat electrolyte losses, potassium and magnesium losses in urine were unaffected by dietary intervention, while sodium and chloride losses were dramatically different. Daily acclimation trials apparently depressed urine potassium excretion during both diets, but magnesium excretion was not significantly altered.

Table V incorporates data from Tables III and IV, details dietary intake, and describes whole-body balance during both diets. In terms of differences between electrolyte intake and losses, the nine subjects incurred a mean accumulation of sodium as a result of the high sodium diet ($+916\text{ mEq/8 days}$), and a mean sodium deficit while consuming the low Na^+ diet (-230.4 mEq/8 days). Whole-body potassium balance, however, was very similar for both diets ($+137.6\text{ mEq/8 days}$ vs $+113.6\text{ mEq/8 days}$).

Figure 4 illustrates the results of hematological measurements. Plasma volume changes which accompanied the consumption of the high Na^+ diet were significantly greater than the low Na^+ diet on D4 ($+16.3\%$ vs $+3.0\%$). Interestingly, this plasma volume expansion occurred in four subjects, in spite of body weight decreases. By D9, this large difference was not significant, and both diets were associated with plasma volumes which were significantly larger than PRE values. Figure 4 also presents data concerning two factors which have been theoretically linked to plasma volume expansion--plasma osmolality and plasma protein. Significant differences between diets were found on D4, as plasma osmolality increased and plasma protein concentration decreased. Total circulating plasma protein (g) demonstrated no change on D4. No significant differences between diets were seen on D9. Within-diet differences appeared in plasma protein--as a decrease on D4 and an increase on D9--and in total circulating protein on D9.

The four plasma electrolytes presented in Figure 5 contributed approximately 80% of the osmotically active particles associated with total plasma osmolality in Figure 4 (27). During acclimation trials, plasma potassium concentration remained unchanged (when compared to D1), but the high Na^+ and the low Na^+ diets exhibited significantly different D4 plasma K^+ levels.

Sodium and chloride concentrations also exhibited between-diet differences on D4. However, in contrast to the stability of plasma potassium levels, within-diet increases of plasma sodium, chloride and magnesium were noted on both D4 and D9 of the high Na⁺ diet.

The mean values for total water and electrolyte concentrations in muscle tissue (expressed per 100g of fat-free solid) are presented in Figure 6. It is readily apparent that the dietary treatments produced no statistically significant between-diet differences, throughout all acclimation trials. In fact only muscle water, sodium, chloride and magnesium levels on D9 of the low sodium diet were significantly altered, when compared to PRE levels. Although the magnesium concentration decreased, it is unlikely that this change was physiologically significant.

Discussion

This investigation examined the influence of two levels of dietary Na⁺ intake on the intramuscular and extracellular K⁺ content of nine unacclimatized men who performed eight days of exercise in a hot environment. Electrolyte losses in sweat and urine, plasma electrolyte concentrations, and muscle water and electrolytes were measured during acclimation to assess the potential for ion depletion or hypohydration.

Table V indicates that the dietary treatments were successful in altering whole body stores of Na⁺. Assuming that these subjects had 41 mEq Na⁺ and 47 mEq K⁺ per kg body weight (10) prior to the first treatment, the mean total body stores of these electrolytes were 2948 mEq Na⁺ and 3379 mEq K⁺. The total body Na⁺ stores increased 31.1% (+916 mEq/8 days) when subjects ingested the high Na⁺ diet for eight days, and decreased 7.8% (-230.4 mEq/8 days) during consumption of the low Na⁺ diet. Whole body K⁺ stores increased 4.1% and 3.4% during the high Na⁺ and low Na⁺ diets, respectively. Therefore, dietary Na⁺ intake had essentially no impact on total body K⁺ stores.

It has been hypothesized that the aldosterone-mediated conservation of sodium during training in the heat results in whole body K⁺ depletion, which in turn leads to rhabdomyolysis, defective glycogen synthesis, or heatstroke (12). In the present investigation, urinary K⁺ losses (Table IV) were depressed during both dietary treatments. In addition, total K⁺ excretion in sweat (Table III) remained stable throughout the eight day acclimation period. Because Na⁺ concentration in sweat and urine were vastly different under the influence of high Na⁺ and low Na⁺ diets (Tables III and IV), it can be concluded that the conservation of Na⁺ in urine and sweat had little effect on the physiological mechanisms regulating K⁺ excretion. The stability of total plasma

K⁺ levels further supports this conclusion. The significant difference between diets on D4 (Figure 5) was the result of an isosmotic increase in total K⁺, because the plasma K⁺ concentration (not shown) was not significantly affected by the dietary-acclimation protocol ($p > 0.05$). Muscle biopsy analyses of K⁺ (Figure 6) also were not significantly altered by the ingestion of either diet or by eight days of exercise in the heat.

The urinary K⁺ conservation during both diets (Table IV) is difficult to reconcile in light of the fact that low Na⁺ diets theoretically induce K⁺ excretion. It is important to recognize, however, that the K⁺ homeostatic mechanism is not perfect. Changes in intrarenal physical factors may over-compensate for the hormonal changes induced by the two Na⁺ diets (28). The K⁺ conservation mechanism mentioned above has not been clearly described, yet acclimation studies have reported similar Na⁺ conservation and K⁺ stability in urine or sweat (29, 30, 31).

The present investigation may be criticized on the grounds that subjects exercised for only 90 minutes in the heat, compared to continual exposure to heat in desert living. Harsh desert environments theoretically stimulate larger K⁺ deficits. We believe that this criticism is unjustified, for two reasons. First, if a daily sweat loss of 12 liters is assumed (32) and if maximal urinary and sweat excretion levels were 68.8 mEq K⁺/day and 5.45 mEq K⁺/liter (present investigation), the maximal K⁺ losses of desert living could be offset by a diet which contained 134 mEq K⁺/day. This "worst case" requirement is not unreasonable, considering the fact that three meals of U.S. military C rations provide up to 129 mEq K⁺/day without additional salting (33). Only minor dietary supplementation would be required to remain in K⁺ balance. Second, the metabolic rate of exercise performed during this investigation (44.6-49.7% V_O₂ max) is equivalent to the workload chosen by 12 soldiers who self-paced hard work rates over four different terrains carrying a variety of external loads (34). We believe that the work intensity maintained by subjects in the present investigation was representative of hard outdoor work.

Previous reports of whole body K⁺ deficits contain apparent methodological errors (12,16). The first study (16) failed to verify ⁴²K estimates of whole body K⁺ by measuring the balance of K⁺ and its excretion in sweat, urine or feces. Costill (13) has calculated that the men in that study would have been required to produce over 76 liters of sweat in 4 days--nearly 50% of their total body water--to reach the reported deficits. The latter study by Knochel (12) also reported large K⁺ deficits after 4 days. Interestingly, the net loss of 40 mEq K⁺/day (⁴²K estimates) was accompanied by a dietary intake of 106 mEq K⁺/day, a sweat loss of 37 mEq K⁺/24 hr, and a urinary excretion of 64 mEq K⁺/24 hr. These last three factors would indicate a net gain of 5 mEq K⁺/24 hr

(12, pp. 176-178). Malhorta et al have also reported K⁺ depletion in six males during 4 days of heat exposure (35). Procedural difficulties weaken their argument, however. Urinary and sweat volumes were not measured, and values from other studies were used to formulate conclusions.

Another consideration involves the investigations which have demonstrated the ability of the human body to maintain stable intramuscular K⁺ levels under a variety of conditions. These include prolonged exercise (10), dehydration (36), static maximal voluntary contractions of quadriceps group (37), repeated days of cycling in a hot environment (38), and repeated days of exercise coupled with a diet low in K⁺ (13).

Sodium Excretion

The total urinary sodium output (Table IV) was reduced by 48% after one day on the low Na⁺ diet. The kidneys had maximally conserved sodium by D3 and D4. During the high Na⁺ diet, however, initial urinary Na⁺ levels were similar to the low Na⁺ diet, yet rose 24-58% on all remaining days. Table III illustrates that the total sweat Na⁺ losses decreased by 26% on D4 and by 40% on D8, with the low Na⁺ diet. Similar to the urine data above, the high Na⁺ diet resulted in increased Na⁺ and sweat losses (+30% on D4 and 41% on D8). Chloride excretion trends in urine and sweat during acclimation verified the Na⁺ excretion patterns described above.

The kidneys apparently conserved sodium (low Na⁺ diet) or wasted sodium (high Na⁺ diet) via the renin-angiotensin-aldosterone mechanism. Convertino, Greenleaf and Bernauer (39) have concluded that this mechanism enhances fluid retention during acclimation and leads to hypervolemia. It is tempting to offer a similar explanation for sweat gland conservation (or wasting) of Na⁺, but this link has not been substantiated.

Muscle Constituents

The D9 muscle water and electrolyte data (Figure 6) can be interpreted in two ways. First, these increases can be treated as a movement of sodium into muscle tissue which resulted in an osmotic increase of muscle water. The movement of chloride into, and the movement of Mg⁺⁺ out of, muscle tissue would be necessary to maintain ionic neutrality. Yet, there are unmeasured anions and cations (e.g. Ca²⁺, HCO₃⁻, PO₄⁻²), protein molecules and phosphate compounds which contribute to intracellular ionic neutrality. Second, these data may be viewed as artifact, due to minor external contamination. Bergstrom (25) and Costill (10) have cautioned that unknown contamination of extracellular electrolytes may exist with the muscle analysis technique used in the present investigation. The loss of Mg⁺⁺, which is primarily an intracellular species and which is less susceptible to contamination, would indicate that an actual decrease in D4 intramuscular Mg⁺⁺ had occurred, however.

Acclimation Adaptations

Statistically significant decreases in mean heart rate and rectal temperature were observed from D3 through D8 of acclimation trials (when compared to D1). These adaptations have been well documented by previous investigators. Sweat responsiveness per degree centigrade increased from D1 to D8 in nearly all exercise trials, in support of the work of Nadel and colleagues (40). Sweat rate exhibited no change during acclimation. This was not surprising because the rate of sweating in the present study was sub-maximal (470-550 ml/m²·hr) and the increase in sweat rate which accompanies acclimation occurs at near-maximal levels (41). Previous investigations which have reported no increase in sweat rate during acclimation have involved low work intensities and relative low sweat rates (32,41,42,43).

Significant differences between the high and low Na⁺ diets were seen only on D3-D5 (heart rate) and D3-D6 (core temperature); by D8, these differences were not evident. An earlier investigation conducted by Taylor *et al* (4) similarly observed higher pulse rates and rectal temperatures in men who had consumed a "low" salt diet (138 mEq Na⁺/day), when they were compared to men on moderate and high salt diets. Concurrently, the plasma volume expansion (Figure 4) exhibited a significant between-diet difference on D4 which was not seen by D9. Sweat responsiveness (ml/hr.°C) was lower in the low Na⁺ diet in 6 out of 8 trials. Thus, 4 days of ingesting the low Na⁺ diet apparently reduced the thermal conductance capacities of these subjects. This was evidenced in a delayed plasma volume expansion, sweat responsiveness decrease, higher rectal temperature, and higher heart rate. The increased heart rate (D3-D5) may have been due to a lower stroke volume, increased core temperature, or a redistribution of cardiac output to cutaneous vascular beds (44).

The difference in plasma volume expansion between diets is of considerable interest (Figure 4). The D4 plasma volume expansion (high Na⁺ diet) was apparently the result of an isosmotic increase in total circulating electrolytes; the plasma osmolality did not increase but plasma volume did. Values for plasma Na⁺, K⁺, Cl⁻ and Mg⁺² (Figure 5) increased in harmony with this interpretation. In contrast, a relatively stable total circulating protein (+8.7g) and a decreased plasma protein concentration (-0.8g%) indicate that protein movement played a minor role in the D4 high Na⁺ diet plasma volume expansion. Assuming that each gram of protein "binds" 14-15ml of water (39,45,46), the 8.7g increase in total plasma protein should have increased the plasma water by 121.8-130.5ml. The actual increase in mean plasma volume from D1 to D4 was 540ml.

The importance of dietary sodium intake as a factor in plasma volume expansion cannot be underestimated. This fact has apparently been ignored in protocol design since 1971, when Smiles and Robinson (18) stated that plasma volume expansion during acclimation occurred only when subjects were in electrolyte and water balance. Few subsequent studies involving plasma volume expansion have controlled

dietary Na⁺ intake or have reported the electrolyte status of their subjects. Typically, acclimation does not alter red cell volume (39), but because Smiles and Robinson utilized only hematocrit to estimate plasma volume shifts, changes in plasma Na⁺ concentration may have had osmotic effects on red cell volume, which would then have contributed to measurement errors. The present investigation estimated plasma volume changes using both hemoglobin and hematocrit (22), reducing the error of estimating plasma volume changes potentially resulting from fluctuations in red cell volume (23).

In summary, the ingestion of two widely different levels of Na⁺ had no significant effect on K⁺ levels in urine, sweat, or muscle tissue. Plasma levels of total K⁺ increased (D4), but this increase was isosmotic. In spite of the large differences in dietary Na⁺ content and heavy sweating, subjects were able to maintain a positive K⁺ balance with a normal intake of K⁺ (80 mEq K⁺/day). Heart rate, rectal temperature, plasma volume and sweat sensitivity data indicated that these subjects transferred heat from the core of the body to the environment less effectively when they were in negative Na⁺ balance (low Na⁺ diet). This investigation also underscored the importance of dietary Na⁺ control during future investigations which involve measurements of physiological adaptations to acclimation, especially plasma volume.

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TABLE I
SUBJECT CHARACTERISTICS (n = 9)

	<u>Age</u> (yr)	<u>Height</u> (cm)	<u>Weight</u> (kg)	<u>Body fat</u> (%)	$\dot{V}O_2$ max (l/min)	$\dot{V}O_2$ max (ml/kg/min)
MEAN	24.7	177.2	71.9	11.5	3.70	51.1
\pm SE	± 1.6	± 1.9	± 3.3	± 0.9	± 0.20	± 1.1

TABLE II

SWEAT RATE($\text{ml/hr}\cdot\text{m}^2$) AND SWEAT RESPONSIVENESS($\text{ml/hr}/^{\circ}\text{C}_{\text{rectal}}$).

BOTH DIETS INVOLVED 9 SUBJECTS AND REPRESENT MEAN \pm SE VALUES.

Dietary Treatment	DAYS OF ACCLIMATION							
	D1	D2	D3	D4	D5	D6	D7	D8
	SWEAT RATE ^①							
High Na+	530 +40	470 +50	480 +30	490 +40	530 +40	500 +20	550 +30	550 +30
Low Na+	500 +60	470 +50	520 +80	530 +40	550 +50	550 +40	540 +40	510 +30
	SWEAT RESPONSIVENESS							
High Na+	820 +130	820 +100	810 + 80	910 +150	930 + 70	840 + 50	900 + 90	1060 +110
Low Na+	880 + 70	610* + 90	770 +100	730 +110	860 +130	850 + 80	840 +100	890 + 80

① - based on mean skin surface area of 1.88m^2

◆ - diets significantly different ($p < .05$)

* - this day is significantly different from day1 ($p < .05$)

TABLE IIIa

SWEAT ELECTROLYTE LOSSES (total mEq/90 min)
DURING ACCLIMATION TRIALS

Measurement	Diet	DAYS OF ACCLIMATION		
		D1	D4	D8
Na+	HIGH Na+	61.2 +7.9	79.51 +12.7	86.53* +16.3
	LOW Na+	63.2 +7.4	46.9 +9.6	37.8* +3.2
Cl-	HIGH Na+	50.3 +8.1	69.8 +11.3	78.1* +14.1
	LOW Na+	64.8 +15.0	39.7* +8.5	44.5* +11.9

* - This day signif. different from Day 1 ($P < .05$)

◆ - Diets signif. different ($P < .05$)

TABLE IIIb

SWEAT ELECTROLYTE LOSSES (total mEq/90 min)

DURING ACCLIMATION TRIALS

Measurement	Diet	DAYS OF ACCLIMATION		
		D1	D4	D8
K+	HIGH Na+	6.61 + .74	7.06 + .97	7.18 + .69
	LOW Na+	7.93 + .63	8.43 + .76	8.42 + .79
Mg++	HIGH Na+	1.88 + .17	1.68 + .14	1.62 + .19
	LOW Na+	2.04 + .37	1.79 + .16	1.59 + .19

* - This day signif. different from Day 1 ($P < .05$)

◆ - Diets signif. different ($P < .05$)

TABLE IVa

DAILY URINE VOLUME (ml/24 hr) AND ELECTROLYTE LOSSES (total mEq/24 hr), (Mean \pm SE)										
Measurement	Diet	CONTROL ¹ VALUE	D1	D2	D3	D4	D5	D6	D7	D8
Urine Volume	HIGH Na ⁺		912 [*] +84.42	1430 +210	1294 +129	1123 +107	1536 +228	1112 +117	1065 +141	1096 +126
	LOW Na ⁺	1268 +146					◆			
Na ⁺	HIGH Na ⁺		1218 +196	1018 +133	824 [*] +105	862 [*] +95	932 +155	1068 +128	1019 +165	1229 +213
	LOW Na ⁺	171.3 +17.7	171.9 +24.7	257.0 [*] +46.3	270.6 [*] +35.4	212.7 +30.7	256.8 [*] +28.8	242.1 [*] +53.7	235.0 [*] +36.6	214.8 +31.7
Cl ⁻	HIGH Na ⁺		88.8 [*] +9.6	40.2 [*] +7.9	27.6 [*] +4.2	29.3 [*] +5.4	32.0 [*] +5.3	42.9 [*] +9.3	67.6 [*] +22.6	82.8 [*] +21.5
	LOW Na ⁺	144.8 +14.3	171.2 +26.2	246.3 [*] +43.1	244.7 [*] +31.0	251.4 [*] +36.9	290.7 [*] +22.4	230.5 [*] +24.1	247.3 [*] +38.3	206.5 [*] +27.4
	HIGH Na ⁺		◆	◆	◆	◆	◆	◆	◆	◆
	LOW Na ⁺		89.2 [*] +12.1	43.6 [*] +9.0	25.8 [*] +4.3	28.0 [*] +5.5	29.5 [*] +4.9	41.3 [*] +8.4	44.5 [*] +7.9	56.2 [*] +11.4

◆ - diets signif. different (P<.05)

* - this day signif. different from control (P<.05)

** - represents two 24 hr samples per subject, collected 1-4 days prior to second treatment. Subjects consumed their usual civilian diets.

TABLE IVb

DAILY URINE VOLUME (ml/24 hr) AND ELECTROLYTE LOSSES (total mEq/24 hr), (Mean \pm SE)										
Measurement	DIET	CONTROL ^o VALUE	DAYS OF ACCLIMATION							
			D1	D2	D3	D4	D5	D6	D7	D8
Urine Volume	HIGH Na ⁺		912 [*] ± 84.42	1430 ± 210	1294 ± 129	1123 ± 107	1536 ± 228	1112 ± 117	1065 ± 141	1096 ± 126
	LOW Na ⁺	1268 ± 146								
K ⁺	HIGH Na ⁺		1218 ± 196	1018 ± 133	824 [*] ± 105	862 [*] ± 95	932 ± 155	1068 ± 128	1019 ± 165	1229 ± 213
	LOW Na ⁺	56.2 ± 4.2	56.2 ± 7.9	68.8 ± 10.5	51.3 ± 5.1	41.2 ± 6.0	40.0 [*] ± 4.8	42.5 ± 8.9	42.3 ± 5.6	34.2 [*] ± 5.7
Mg ⁺⁺	HIGH Na ⁺		50.1 ± 4.5	54.7 ± 8.9	39.9 [*] ± 2.1	37.0 [*] ± 3.2	40.8 ± 2.5	40.4 [*] ± 5.2	41.7 ± 8.7	49.7 ± 8.5
	LOW Na ⁺	9.1 ± 1.4	9.9 ± 1.4	13.0 ± 2.3	12.0 ± 1.7	10.1 ± 1.2	12.0 ± 0.87	10.8 ± 1.2	10.7 ± 1.2	12.5 ± 1.6
	HIGH Na ⁺		7.7 $\pm .7$	10.7 ± 1.2	8.2 $\pm .7$	9.2 ± 0.9	8.7 ± 1.1	9.2 ± 1.2	8.9 ± 0.9	11.2 ± 1.5
	LOW Na ⁺									

◆ - diets signif. different ($P < .05$)

* - this day signif. different from control ($P < .05$)

o - represents two 24 hr samples per subject, collected 1-4 days prior to second treatment. Subjects consumed their usual civilian diets.

TABLE IV
WHOLE BODY SODIUM AND POTASSIUM BALANCE CONSIDERING
URINE LOSS, SWEAT LOSS AND DIETARY INTAKE (total mEq/24 hr)

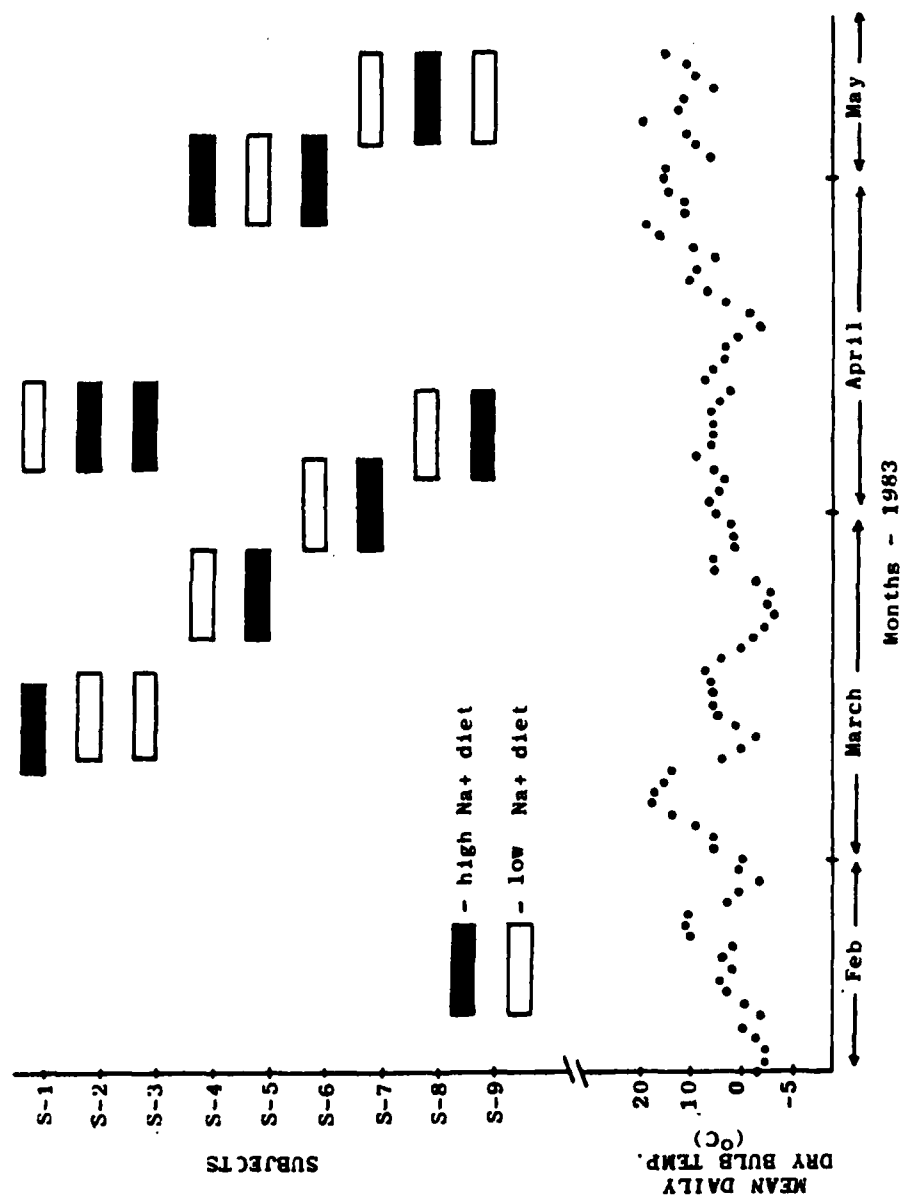
Diet	Source of Loss	Control	DAYS OF ACCLIMATION			Three Day Mean Loss*	Daily Dietary Intake	Mean Daily Balance*	Eight Day Balance*
			D1	D4	D8				
<u>SODIUM BALANCE</u>									
HIGH Na+	Urine	171.3	171.9	212.7	214.8	284.5	399	+114.5	+916.0
	Sweat		61.2	79.5	86.5				
	TOTAL		233.1	292.2	301.3				
LOW Na+	Urine	171.3	88.8	29.3	82.8	126.8	98	- 28.8	-230.4
	Sweat		63.2	51.6	37.8				
	TOTAL		152.0	80.9	120.6				
<u>POTASSIUM BALANCE</u>									
HIGH Na+	Urine	56.2	56.2	41.2	34.2	62.8	80	+ 17.2	+137.6
	Sweat		6.6	7.1	7.2				
	TOTAL		62.8	48.3	41.4				
LOW Na+	Urine	56.2	50.1	37.0	49.7	65.8	80	+ 14.2	+113.6
	Sweat		7.9	6.4	8.4				
	TOTAL		58.0	45.4	58.1				

* - includes correction for electrolyte loss in feces: 1.5mEq Na+/day and 4mEq K+/day (ref. 26).

LEGEND OF FIGURES

- Figure 1 Order of 8 day dietary treatments and mean environmental dry bulb temperatures during acclimation trials.
- Figure 2 Schedule of exercise bouts and physiological testing during the two dietary regimens.
- Figure 3 Physiological measurements during acclimation trials (mean \pm SE, n=9).
- Figure 4 Mean (+ SE) plasma volume osmolality and protein values before and after 4 and 9 days of heat acclimation.
- Figure 5 Selected plasma electrolytes (total mEq).
- Figure 6a Muscle water and sodium contents before and during heat acclimation.
- Figure 6b Muscle water and potassium contents before and during heat acclimation.

FIGURE 1



Order of 8 day dietary treatments and mean dry bulb temperatures during acclimation trials.

FIGURE 2

Variable	Control	Treatment days								
		1	2	3	4	5	6	7	8	9
90 min. exercise* (40 C, 23.5% RH)		X	X	X	X	X	X	X	X	
24 hr. urine output			X	X	X	X	X	X	X	X
Sweat washdown		X			X				X	
Blood sample	X				X					X
Blood total protein	X				X					X
Blood osmolality	X				X					X
Blood hemoglobin and hematocrit	X				X					X
Plasma volume (T-1824)	X				X					X
Muscle biopsy	X				X					X

X - sample taken

* - includes heart rate, rectal temperature,
respiratory exchangeSchedule of exercise bouts and physiological sampling
during both dietary treatments

FIGURE 3

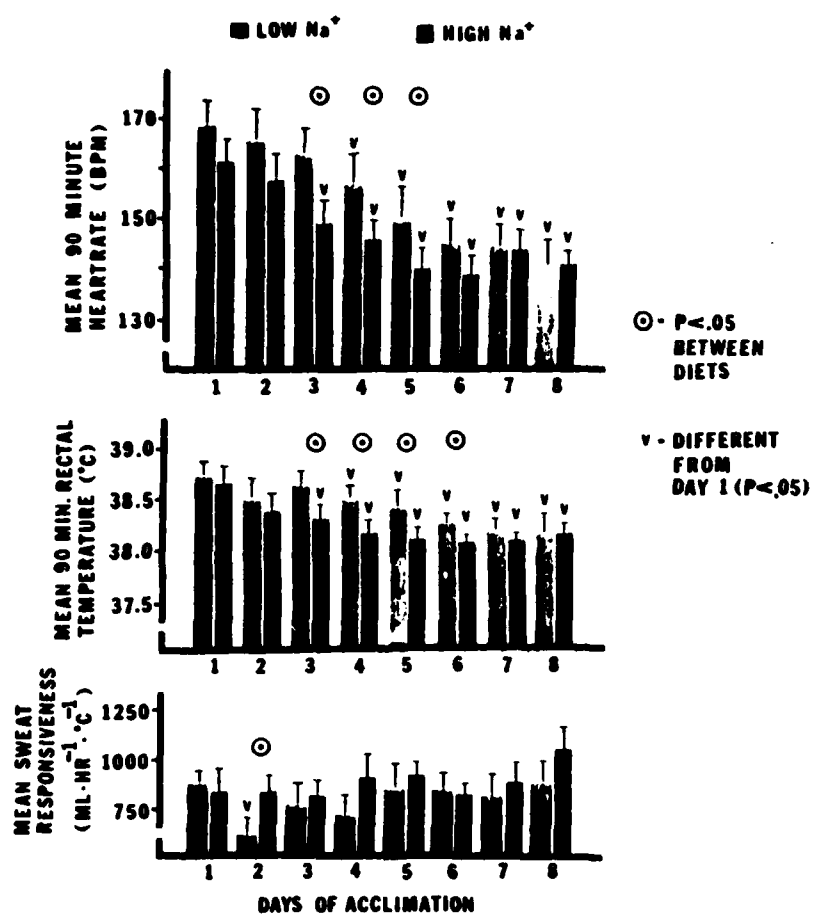


FIGURE 4

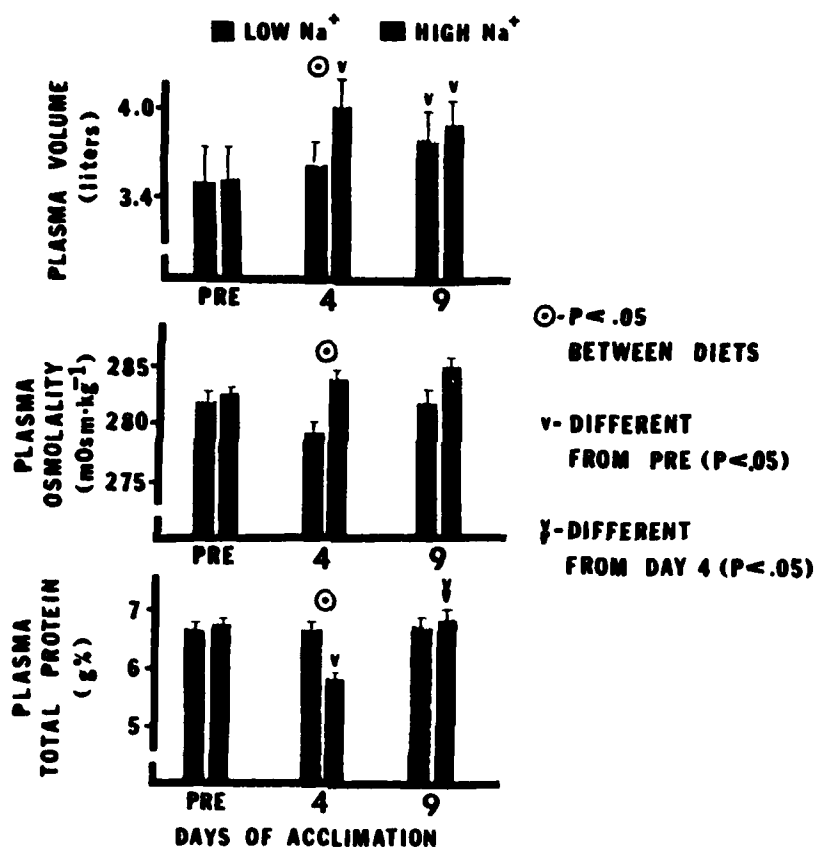


FIGURE 5

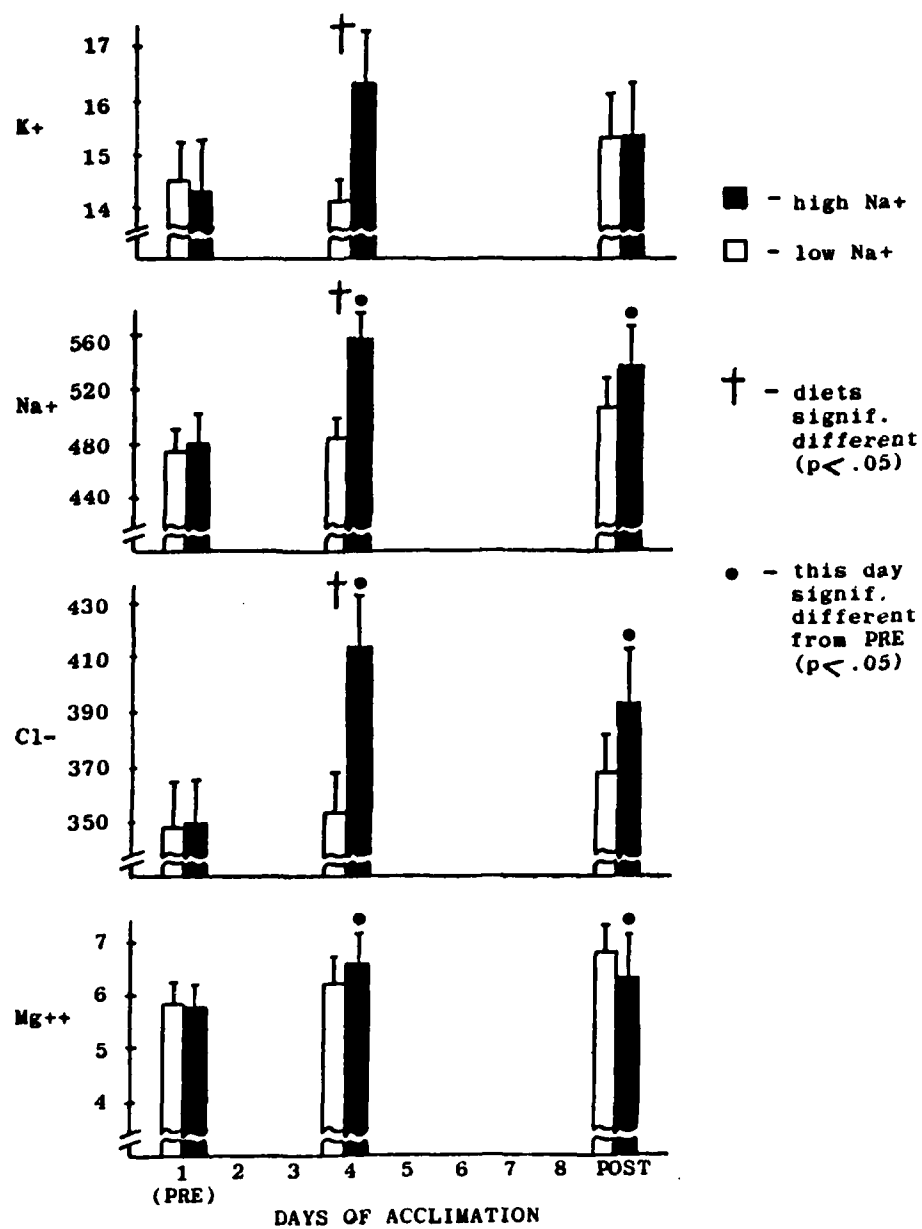
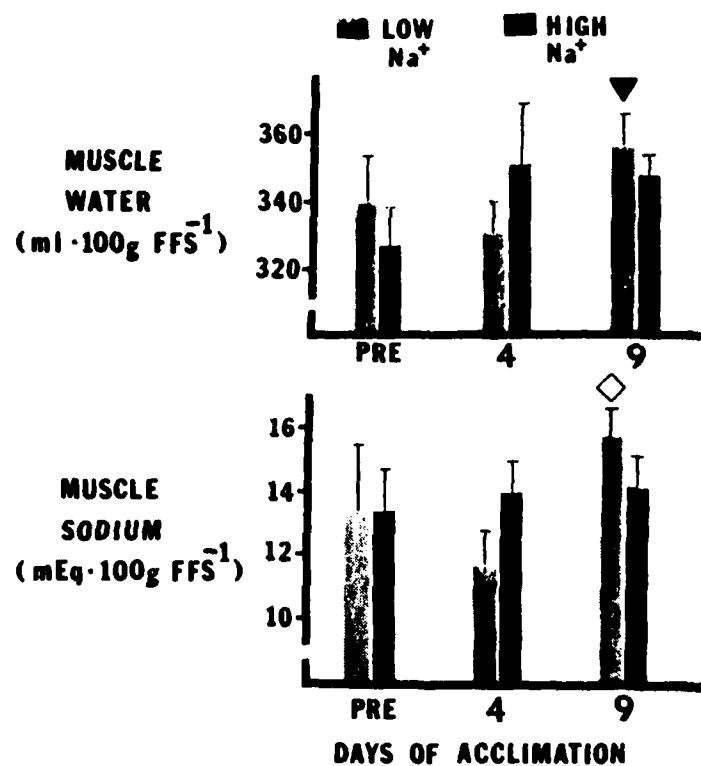


Figure VI - Selected plasma electrolytes (total mEq), mean \pm SE (n = 9)

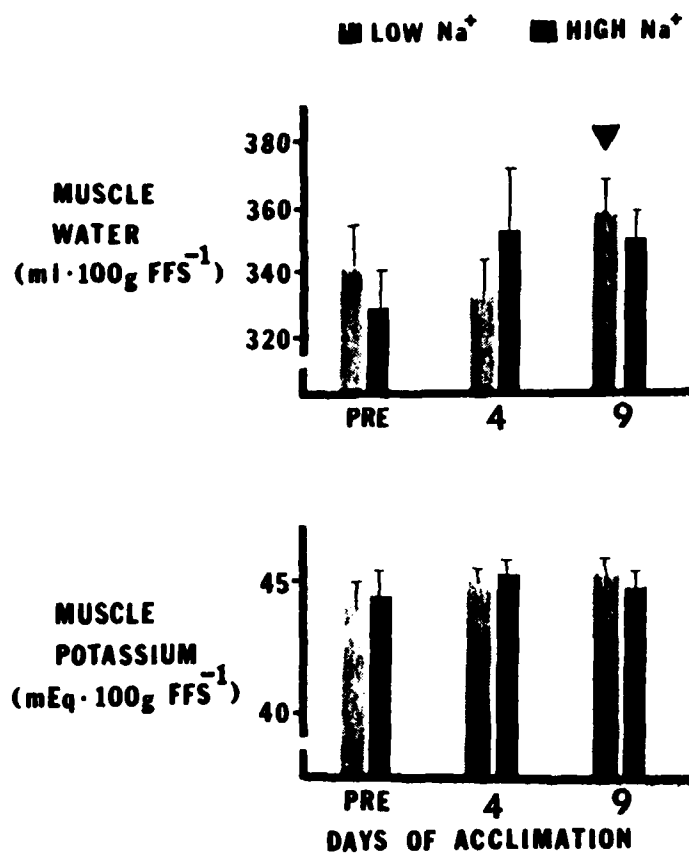
FIGURE 6a



▼ - DIFFERENT FROM PRE & DAY 4 (P < .05)

◊ - DIFFERENT FROM DAY 4 (P < .05)

FIGURE 6b



▼ - DIFFERENT FROM PRE & DAY 4 (P < .05)

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